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Management of the peri-intubation period in patients with pulmonary arterial hypertension and respiratory failure

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Abstract

Purpose of Review The endotracheal intubation of patients with pulmonary arterial hypertension (PAH) in respiratory distress is a highly morbid procedure that can precipitate hemodynamic collapse. Here we review our strategy for confronting this difficult clinical situation.

Recent findings There are no clinical trials that explore best practices in the management of patients with PAH and respiratory failure.

Summary Here we provide a practical approach to respiratory support, inopressor and pulmonary vasodilator selection, hemodynamic considerations, point-of-care ultrasound monitoring, and endotracheal intubation in patients with PAH in respiratory failure.

Keywords Intubation · Respiratory failure · Pulmonary hypertension · Right ventricular failure

Introduction

Respiratory failure requiring endotracheal intubation in patients with pulmonary arterial hypertension (PAH) is a harrowing event that can easily precipitate right ventricular (RV) failure and cardiac arrest [1]. Hospital mortality rates approach 40% in patients with PAH that require mechanical ventilation, a statistic that becomes significantly grimmer in the context of respiratory failure [2, 3]. While there is a paucity of clinical data to draw from, the predominant drivers of hemodynamic collapse relate to the acute reduction in venous return upon the initiation of positive pressure ventilation [4], the negative inotropic effects of respiratory acidosis and administered anesthetics [5], and an increase in RV afterload caused by hypoxemia, acidosis and increased intrathoracic pressure [6, 7••]. Furthermore, the concept of ventricular preload is dynamic in decompensated PAH, as there is increased ventricular interdependence owing to increased RV size, thickness, and pressure [8]. As such, this population can exhibit large (and at times opposing) swings in right and left ventricular preload during the peri-intubation period. A comprehensive understanding of the drivers of a specific patient's pulmonary hypertension and their degree of RV dysfunction is needed in order to predict the likely response to a particular intervention and the initiation of positive pressure ventilation [9•]. Given the myriad challenges faced by the RV in the setting of respiratory failure, the peri-intubation period requires foresight, preparation, and efficient implementation of a pre-defined intubation plan to increase the likelihood of a smooth transition to mechanical ventilation. Here we review our strategy for confronting this difficult clinical scenario (Fig. 1).

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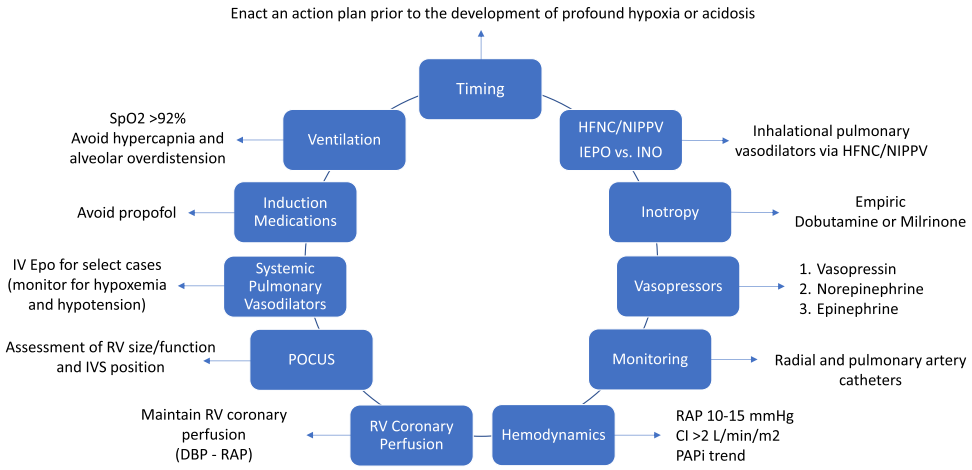

Fig. 1 Management of the Peri-intubation Period in Patients with Pulmonary Arterial Hypertension and Respiratory Failure. Legend: Comprehensive strategy for transitioning patients with pulmonary arterial hypertension and respiratory failure to invasive mechanical ventilation. CI: cardiac index. DBP: diastolic blood pressure. HFNC: high-flow nasal cannula. IEPO: inhaled epoprostenol. INO: inhaled nitric oxide. IV Epo: intravenous epoprostenol. IVS: interventricular septum. NIPPV: non-invasive positive pressure ventilation. PAPi: pulmonary artery pulsatility index. POCUS: point-of-care ultrasound. RAP: right atrial pressure. RV: right ventricle. SpO2: oxygen saturation

Timing

A patient's stability, rate of cardiopulmonary deterioration, response to rescue interventions and resource availability will dictate how many of the concepts discussed in this article will be logistically feasible. Perhaps most importantly, intubation should not be deferred to the point of profound respiratory distress and impending cardiac arrest for the sake of exhaustive pre-intubation planning, as resuscitative outcomes in this population are extremely poor [10, 11]. In patients with progressive or end stage PAH in whom there is no reversible cause of respiratory failure or definitive therapy, not offering invasive mechanical ventilation is often appropriate as it will likely be medically ineffective and may hasten decompensation. We recommend thoughtful goals of care conversations with the help of a palliative care team in any patient with PAH at risk of developing respiratory failure [12].

Non-Invasive Respiratory Support

In general, respiratory support should be titrated to achieve normocapnia and a pulse oximeter saturation > 92% [11, 13]. Consider using high-flow nasal cannula or non-invasive positive pressure ventilation to oxygenate, improve ventilation, and reduce work of breathing while preparing for intubation. Additionally, non-invasive respiratory support is necessary to deliver inhalational pulmonary vasodilators such as epoprostenol or nitric oxide [14].

When administering non-invasive positive pressure ventilation, it's important to remember that the relationship between pulmonary vascular resistance and lung volume is a U-shaped curve, with both extremes of lung volume causing increased RV afterload [15•]. Under conditions of low lung volumes, areas of atelectasis form and the alveolar arterioles in those regions vasoconstrict to direct blood to aerated regions to prevent ventilation-perfusion (VQ) mismatch. At the other extreme, high lung volumes increase RV afterload by causing pneumatic compression of the pulmonary vasculature. In practice, we recommend starting with minimal respiratory support settings (e.g., positive end-expiratory pressure of 5 cmH2O) with cautious titration as needed to meet oxygenation, ventilation, and hemodynamic goals.

RV Contractility

We initiate inotropes early to support the RV throughout the peri-intubation period. When choosing an inotrope, consider the hemodynamic and pharmacokinetic properties of the potential agent to inform your decision. The pulmonary vasodilator effects of milrinone may make it the superior inotrope for patients with pulmonary hypertension [16]. However, it causes more peripheral vasodilation and thus hypotension and has a longer time to initial hemodynamic effect as compared to dobutamine (30 vs. 1 min) [17, 18] and can accumulate in patients with renal impairment [19]. As such, patient stability and the urgency of intubation should help guide agent selection. In general, we will start

dobutamine 5 mcg/kg/min at the first signs of respiratory distress. In patients with hypotension, epinephrine may be the preferred inotropic agent. Additionally, as acidosis is a significant depressant of myocardial contractility we recommend active acid–base management by means of ventilatory support, renal replacement therapy, or temporary bicarbonate supplementation if respiratory compensation is inadequate, targeting a pH of 7.3–7.4 [20].

Vasopressors

Our practice is to use vasopressin as the first-line vasopressor, as it's a potent systemic vasoconstrictor with minimal impact on the pulmonary vasculature [21]. Norepinephrine is our standard second-line vasopressor, and epinephrine the third. Phenylephrine should be avoided due to its potent pulmonary vasculature vasoconstrictor effects [14, 22]. In patients with significant right ventricular dysfunction, we anticipate hemodynamic swings throughout the peri-intubation period and will commonly start vasopressin 0.04 Units/min prior to intubation and have a second- and third-line vasopressor prepared for immediate use (i.e., having the bag spiked and hung, tubing primed and connected to the patient). In particularly unstable patients consider having bolus dose vasopressin (0.5–1.0 unit/mL), norepinephrine (16 mcg/mL), and/or epinephrine (10 mcg/mL) on hand. We typically start with bolus volumes of 0.5–1.0 mL at the above concentrations and double the bolus injection amount if the hemodynamic response is inadequate after 30 seconds of circulation. With every bolus requirement we concurrently increase the infusion rate of the particular agent to ensure that as the bolus effect wanes the infusion will maintain the desired steady state concentration.

Pulmonary Vasodilators

We recommend close collaboration with a PAH expert when initiating and titrating pulmonary vasodilators. In the setting of acute respiratory failure, oral and parenteral pulmonary vasodilators can cause worsening hypoxia and hypercapnia by exaggerating shunting through areas of lung that are not participating in gas exchange (i.e., systemically acting pulmonary vasodilators attenuate the lung's ability to maintain VQ matching via adaptive hypoxic pulmonary vasoconstriction). Patients on chronic systemic pulmonary vasodilators may develop hypoxia out of proportion to a given pulmonary insult due to this phenomenon, and may require careful adjustment of PAH therapies, including temporary dose reduction or discontinuation. However, sudden withdrawal of PAH specific therapies, specifically parenteral prostacyclins, may precipitate acute RV failure, so extreme caution and close monitoring is required when adjusting these medications. Inhaled pulmonary vasodilators (e.g., inhaled epoprostenol and nitric oxide), in contrast, only act in areas of the lung that are effectively participating in alveolar gas exchange. As such, inhaled pulmonary vasodilators may improve VQ matching in addition to reducing RV afterload [14].

In carefully selected patients with group 1 PAH and severe RV dysfunction, the hemodynamic benefits of initiating parenteral prostacyclin may outweigh the risk of exaggerating pulmonary shunting. In these cases, we typically choose intravenous epoprostenol as it is a potent pulmonary vasodilator with a short half-life on the order of 3–6 min [23]. Dosing should start at 1–2 ng/kg/min and can be uptitrated by 1–2 ng/kg/min every 15–30 min as tolerated [7••]. Frequent dose-limiting effects to monitor in addition to worsening pulmonary shunting are hypotension, flushing, jaw pain, myalgias, diarrhea, nausea, vomiting and headache.

Hemodynamics

Invasive hemodynamic monitoring with arterial and pulmonary artery catheters are invaluable in navigating the management of critically ill patients with PAH. Our strategy is to use trends in the right atrial pressure (RAP), systolic pulmonary pressure, pulmonary artery pulsatility index (PAPi; [pulmonary systolic pressure minus pulmonary diastolic pressure] divided by RAP]), and cardiac index to guide inotropic and vasoactive therapies. In general, we use the RAP, cardiac index and PAPi as invasive indicators of RV reserve, and a RAP greater than 15 mmHg, cardiac index less than 2 L/min/m2 or fall in PAPi as a trigger for additional intervention. Notably, the PAPi is inherently high in patients with PAH, and the lower cut-point associated with mortality (PAPi < 5.3) is considerably higher than the values described for its more common utility as a marker of RV failure in the setting of acute myocardial infarction and after left ventricular assist device implantation [24–26]. Furthermore, given that the PAPi has not been studied in acute decompensated PAH, we find that trending an individual's PAPi is more telling of RV—pulmonary arterial coupling than is using a binary cut-point value.

When optimizing preload, it is hard to predict the optimal filling pressure required to maximize the Frank-Starling mechanism of a hypertrophied RV, but 10–15 mmHg is a reasonable target. If the RAP exceeds 15 mmHg, we pursue modes of volume removal with diuretic therapy, slow continuous ultrafiltration (SCUF), or continuous veno-venous hemofiltration (CVVH) as appropriate. In rare cases where the RAP is less than 5 mmHg, we may consider cautiously administering small amounts of fluid (250–500 mL) to compensate for the

sudden reduction in venous return expected during the transition to positive pressure ventilation. Additionally, it is worth noting that significant increases in RV preload can arise when laying patients supine for intubation or placing in Trendelenburg for central line or pulmonary artery catheter placement, and such changes in position should be performed cautiously.

RV Coronary Perfusion

As pressure in the RV approaches near-systemic blood pressure, coronary blood flow to the RV myocardium is limited to the diastolic phase of the cardiac cycle, similar to the pattern of coronary blood flow in the healthy left ventricle (LV) [27, 28]. Under this condition, the RV coronary perfusion pressure (CPP) can be approximated as the pressure gradient between the systemic diastolic blood pressure (DBP) and the RV diastolic pressure. By using the RAP to approximate the RV diastolic pressure, this equation simplifies to the systemic diastolic blood pressure minus the RAP (i.e., RV CPP = DBP minus RAP). RV ischemia and rapid deterioration can occur with transient reductions in RV CPP, even in the absence of significant coronary artery disease. As such, maintaining an adequate gradient between DBP and RAP is paramount to avoid a hemodynamic downward spiral (hypotension causing ischemia causing more hypotension, etc.) [29]. This is achieved primarily by maintaining a patient's DBP throughout the entire intubation process via the proactive use of vasoactive and inotropic medications, as discussed above.

Point-of-Care Ultrasound (POCUS)

Describing the various quantitative echocardiographic metrics of RV function are beyond the scope of the review [30]. However, a basic POCUS assessment of RV size and function (i.e., mild, moderate, or severe) and interventricular septal (IVS) position can greatly help to tailor therapy. RV dilation and bowing of the IVS toward the LV places both ventricles at a mechanical disadvantage and can limit left ventricular filling to the point that the LV becomes the cardiac output limiting ventricle [31]. It is helpful to break this concept of RV failure related exaggerated ventricular interdependence into its component cardiac cycle phases. In situations where the IVS is shifted toward the LV in diastole (i.e., a volume overloaded RV), the goal is to decompress the RV via aggressive volume removal, increased inotropy, and RV afterload reduction with pulmonary vasodilators. When the IVS is shifted toward the LV in systole (i.e., a pressure overloaded RV) the strategy is to maintain a systemic systolic pressure greater than the pulmonary systolic pressure using vasopressors and pulmonary vasodilators with less emphasis on volume removal. In either case, regaining neutral IVS positioning will improve cardiac mechanics and hence cardiac output. In the absence of invasive hemodynamics, pulse wave Doppler of the LV outflow tract can be used to obtain a velocity time integral, which can service as a trendable surrogate for stroke volume [32]. Additionally, the POCUS assessment of RAP by means of inferior vena cava size and collapsibility has been validated in patients with pulmonary hypertension [33]. However, this method is less accurate in patients with moderate or severe tricuspid regurgitation and those receiving positive pressure ventilation [30, 34].

Atrioventricular Synchrony

Loss of atrioventricular synchrony is not well tolerated by a failing RV, and therefore we maintain a low threshold to electrically or pharmacologically cardiovert atrial arrhythmias. Amiodarone and digoxin (alone or in combination) are our antiarrhythmics of choice given their relatively stable hemodynamic profile. Notably, digoxin provides a modest increase in inotropy, and slows atrioventricular nodal conduction, but has minimal effect on restoring sinus rhythm [35]. Minimizing arrhythmic triggers through electrolyte repletion, reducing atrial distention via volume removal, treating fever, and ensuring that central venous catheters do not terminate in the right atrium prior to attempts at chemical or pharmacological cardioversion can increase the likelihood of achieving and or maintaining normal sinus rhythm. Similarly, as ventricular arrythmias can quickly prove fatal in this population, we will commonly treat frequent nonsustained ventricular arrythmias with amiodarone and escalate to the addition of lidocaine as needed. Further to this point, in cases where the patient has a pacemaker with an atrial lead, we may attempt to overdrive pace suppress significant ectopy using the AAI mode at a rate between 90–110 BPM.

Intubation

The induction medication regimen should be driven by the agent's pharmacological hemodynamic profile. When performing a Rapid Sequence Intubation (RSI) we recommend some combination of short-acting opioid (e.g., fentanyl 25–100 mcg), benzodiazepine (e.g., midazolam 1–2 mg), and etomidate (0.2–0.3 mg/kg, up to 20 mg), which have minimal direct vasoactive or inotropic effects. Propofol should be avoided given its vasodilatory and negative inotropic profile [4], and we generally avoid ketamine as a primary induction agent as it can increase pulmonary vascular resistance and may be a negative inotrope at high doses in chronically ill patients [36, 37].

Any patient with acute respiratory insufficiency can have marked endogenous catecholamine in circulation and elevated sympathetic tone. Even the most "stable" induction regimen can acutely ablate endogenous catecholamine release, leading to acute hypotension; hence the importance of having continuous infusion and bolus dose vasopressor available.

Induction of general anesthesia for intubation in patients with PAH requires the maintenance of adequate airway patency and rapid transition to positive pressure ventilation via endotracheal intubation. Gathering any intubation history and performing an airway assessment may allow providers to limit unanticipated airway complications, although their predictive utility can be lower than many realize [38, 39]. In patients who are not undergoing RSI, anticipation, recognition and rapid correction of airway obstruction using an oropharyngeal or nasopharyngeal airway is vital to avoid acute hypercarbic acidosis and hypoxemia. The intubation should be performed by the most proficient airway expert available to prevent delays in oxygenation and ventilation, and video laryngoscopy should be favored over direct laryngoscopy to improve the first pass success rate [40]. Optimizing alveolar recruitment with positive end-expiratory pressure to approximate the functional residual capacity (FRC) can help to reduce pulmonary vascular resistance [41]. However, alveolar overdistention during manual bag valve ventilation or when transitioning to the ventilator must be avoided, as pneumatic compression of the pulmonary vasculature can precipitate acute hemodynamic collapse [14].

The risks of induction can be circumvented entirely by utilizing a technique of awake fiberoptic intubation in patients that have the respiratory reserve to tolerate topical anesthesia and airway manipulation [42]. In highly select cases, pursuing awake extracorporeal membrane oxygenation (ECMO) to avoid the need for intubation and possible hemodynamic arrest may be the safest course of action, assuming that a reversible pathology or long-term intervention can be offered to allow for eventual ECMO decannulation.

Conclusion

Here we report our general framework for managing the highly morbid clinical scenario of respiratory failure requiring endotracheal intubation in patients with PAH. If the decision is made to proceed with intubation, the risks may be mitigated by implementing a peri-intubation strategy that incorporates invasive and non-invasive monitoring to provide goal-directed hemodynamic management that is tailored to the patient's physiology. Given the grim prognosis, early goals of care counseling should be encouraged, with the identification of reversible processes (e.g., pneumonia) or definitive therapies (e.g., lung transplant) strongly informing the discussion.

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References

Papers of particular interest, published recently, have been highlighted as: • Of importance •• Of major importance

1. Hoeper MM, Humbert M, Souza R, Idrees M, Kawut SM, Sliwa-Hahnle K, et al. A global view of pulmonary hypertension. Lancet Respir Med. 2016;4(4):306–22.
2. Rush B, Biagioni BJ, Berger L, McDermid R. Mechanical Ventilation Outcomes in Patients With Pulmonary Hypertension in the United States: A National Retrospective Cohort Analysis. J Intensive Care Med. 2017;32(10):588–92.
3. Gopalratnam K, Odeyemi Y, Del Valle KT, Frank RD, Jentzer JC, Gajic O, DuBrock HM. Characteristics and outcomes of critically Ill patients with pulmonary hypertension who undergo endotracheal intubation and mechanical ventilation. J Intensive Care Med. 2023;38(12):1174–82.
4. Hoeper MM, Granton J. Intensive care unit management of patients with severe pulmonary hypertension and right heart failure. Am J Respir Crit Care Med. 2011;184(10):1114–24.
5. Viitanen A, Salmenpera M, Heinonen J. Right ventricular response to hypercarbia after cardiac surgery. Anesthesiology. 1990;73(3):393–400.
6. Lumb AB, Slinger P. Hypoxic pulmonary vasoconstriction: physiology and anesthetic implications. Anesthesiology. 2015;122(4):932–46.
7.•• Barnett CF, O'Brien C, De Marco T. (2022) Critical care management of the patient with pulmonary hypertension. Eur Heart J Acute Cardiovasc Care. 2022;11(1):77–83. **This is a contemporary review on the management of critically ill patients with pulmonary hypertension.**
8. Burkett DA, Slorach C, Patel SS, Redington AN, Ivy DD, Mertens L, et al. Impact of Pulmonary Hemodynamics and Ventricular Interdependence on Left Ventricular Diastolic Function in Children With Pulmonary Hypertension. Circ Cardiovasc Imaging. 2016;9(9).
9.• Humbert M, Kovacs G, Hoeper MM, Badagliacca R, Berger RMF, Brida M, et al. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J. 2022;43(38):3618–731. **Current guidelines for the diagnosis and treatment of pulmonary hypertension.**
10. Hoeper MM, Galie N, Murali S, Olschewski H, Rubenfire M, Robbins IM, et al. Outcome after cardiopulmonary resuscitation in patients with pulmonary arterial hypertension. Am J Respir Crit Care Med. 2002;165(3):341–4.
11. Fullerton DA, McIntyre RC Jr, Kirson LE, St Cyr JA, Whitman GJ, Grover FL. Impact of respiratory acid-base status

in patients with pulmonary hypertension. Ann Thorac Surg. 1996;61(2):696–701.
12. Rhee CL, Cuttica M. Palliative Care and Advance Care Planning in Pulmonary Hypertension. Adv Pulmonary Hypertension. 2018;17(1):15–9.
13. Ventetuolo CE, Klinger JR. Management of acute right ventricular failure in the intensive care unit. Ann Am Thorac Soc. 2014;11(5):811–22.
14. Zamanian RT, Haddad F, Doyle RL, Weinacker AB. Management strategies for patients with pulmonary hypertension in the intensive care unit. Crit Care Med. 2007;35(9):2037–50.
15.• Alviar CL, Miller PE, McAreavey D, Katz JN, Lee B, Moriyama B, et al. Positive Pressure Ventilation in the Cardiac Intensive Care Unit. J Am Coll Cardiol. 2018;72(13):1532–53. **This article describes the effects of positive pressure ventilation on cardiopulmonary physiology.**
16. Eichhorn EJ, Konstam MA, Weiland DS, Roberts DJ, Martin TT, Stransky NB, et al. Differential effects of milrinone and dobutamine on right ventricular preload, afterload and systolic performance in congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol. 1987;60(16):1329–33.
17. Baruch L, Patacsil P, Hameed A, Pina I, Loh E. Pharmacodynamic effects of milrinone with and without a bolus loading infusion. Am Heart J. 2001;141(2):266–73.
18. Thomas RL, Watson D, Marshall LE. Review of intermittent dobutamine infusions for congestive cardiomyopathy. Pharmacotherapy. 1987;7(2):47–53.
19. Shipley JB, Tolman D, Hastillo A, Hess ML. Milrinone: basic and clinical pharmacology and acute and chronic management. Am J Med Sci. 1996;311(6):286–91.
20. Schotola H, Toischer K, Popov AF, Renner A, Schmitto JD, Gummert J, et al. Mild metabolic acidosis impairs the beta-adrenergic response in isolated human failing myocardium. Crit Care. 2012;16(4):R153.
21. Mizota T, Fujiwara K, Hamada M, Matsukawa S, Segawa H. Effect of arginine vasopressin on systemic and pulmonary arterial pressure in a patient with pulmonary hypertension secondary to pulmonary emphysema: a case report. JA Clin Rep. 2017;3(1):1.
22. Rich S, Gubin S, Hart K. The effects of phenylephrine on right ventricular performance in patients with pulmonary hypertension. Chest. 1990;98(5):1102–6.
23. Sitbon O, Vonk Noordegraaf A. Epoprostenol and pulmonary arterial hypertension: 20 years of clinical experience. Eur Respir Rev. 2017;26(143):160055.
24. Ostad S, Sugarman J, Alkhodair A, Liang J, Mielniczuk LM, Hambly N, et al. Association Between the Pulmonary Artery Pulsatility Index and Prognosis in Pulmonary Arterial Hypertension: A Multicentre Study. CJC Open. 2023;5(7):545–53.
25. Mazimba S, Welch TS, Mwansa H, Breathett KK, Kennedy JLW, Mihalek AD, et al. Haemodynamically Derived Pulmonary Artery Pulsatility Index Predicts Mortality in Pulmonary Arterial Hypertension. Heart Lung Circ. 2019;28(5):752–60.
26. Lim Y, Low TT, Chan SP, Lin W, Teo TW, Jang JJ, et al. Does pulmonary artery pulsatility index predict mortality in pulmonary arterial hypertension? ESC Heart Fail. 2021;8(5):3835–44.
27. Prisco SZ, Thenappan T, Prins KW. Treatment Targets for Right Ventricular Dysfunction in Pulmonary Arterial Hypertension. JACC Basic Transl Sci. 2020;5(12):1244–60.
28. van Wolferen SA, Marcus JT, Westerhof N, Spreeuwenberg MD, Marques KM, Bronzwaer JG, et al. Right coronary artery flow impairment in patients with pulmonary hypertension. Eur Heart J. 2008;29(1):120–7.
29. Wanner PM, Filipovic M. The right ventricle-you may forget it, but it will not forget you. J Clin Med. 2020;9(2):432.
30. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr. 2010;23(7):685–713; quiz 86–8.
31. Brookes C, Ravn H, White P, Moeldrup U, Oldershaw P, Redington A. Acute right ventricular dilatation in response to ischemia significantly impairs left ventricular systolic performance. Circulation. 1999;100(7):761–7.
32 Guzzetti E, Capoulade R, Tastet L, Garcia J, Le Ven F, Arsenault M, et al. Estimation of Stroke Volume and Aortic Valve Area in Patients with Aortic Stenosis: A Comparison of Echocardiography versus Cardiovascular Magnetic Resonance. J Am Soc Echocardiogr. 2020;33(8):953-63 e5.
33. Greiner S, Jud A, Aurich M, Hess A, Hilbel T, Hardt S, et al. Reliability of noninvasive assessment of systolic pulmonary artery pressure by Doppler echocardiography compared to right heart catheterization: analysis in a large patient population. J Am Heart Assoc. 2014;3(4):e001103.
34. Kawata T, Daimon M, Nakanishi K, Kimura K, Sawada N, Nakao T, et al. Factors influencing inferior vena cava diameter and its respiratory variation: Simultaneous comparison with hemodynamic data. J Cardiol. 2022;79(5):642–7.
35. Rich S, Seidlitz M, Dodin E, Osimani D, Judd D, Genthner D, et al. The short-term effects of digoxin in patients with right ventricular dysfunction from pulmonary hypertension. Chest. 1998;114(3):787–92.
36. Tweed WA, Minuck M, Mymin D. Circulatory responses to ketamine anesthesia. Anesthesiology. 1972;37(6):613–9.
37. Spotoft H, Korshin JD, Sorensen MB, Skovsted P. The cardiovascular effects of ketamine used for induction of anaesthesia in patients with valvular heart disease. Can Anaesth Soc J. 1979;26(6):463–7.
38. Detsky ME, Jivraj N, Adhikari NK, Friedrich JO, Pinto R, Simel DL, et al. Will This Patient Be Difficult to Intubate?: The Rational Clinical Examination Systematic Review. JAMA. 2019;321(5):493–503.
39. Roth D, Pace NL, Lee A, Hovhannisyan K, Warenits AM, Arrich J, et al. Airway physical examination tests for detection of difficult airway management in apparently normal adult patients. Cochrane Database Syst Rev. 2018;5(5):CD008874.
40. Prekker ME, Driver BE, Trent SA, Resnick-Ault D, Seitz KP, Russell DW, et al. Video versus Direct Laryngoscopy for Tracheal Intubation of Critically Ill Adults. N Engl J Med. 2023;389(5):418–29.
41. Whittenberger JL, Mc GM, Berglund E, Borst HG. Influence of state of inflation of the lung on pulmonary vascular resistance. J Appl Physiol. 1960;15:878–82.
42. Johannes J, Berlin DA, Patel P, Schenck EJ, West FM, Saggar R, et al. A Technique of Awake Bronchoscopic Endotracheal Intubation for Respiratory Failure in Patients With Right Heart Failure and Pulmonary Hypertension. Crit Care Med. 2017;45(9):e980–4.

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